<u>**SCIENTIFIC COMMITTEE ON PLANTS**</u> **SCP/ESFEN/002-FINAL**
6 April 2000

OPINION OF THE SCIENTIFIC COMMITTEE ON PLANTS REGARDING THE INCLUSION OF ESFENVALERATE IN ANNEX 1 OF COUNCIL DIRECTIVE 91/414/EEC CONCERNING THE PLACING OF PLANT PROTECTION PRODUCTS ON THE MARKET

(Opinion expressed by the Scientific Committee on Plants on 17 March 2000)

1. TITLE

OPINION OF THE SCIENTIFIC COMMITTEE ON PLANTS REGARDING THE INCLUSION OF ESFENVALERATE IN ANNEX 1 OF COUNCIL DIRECTIVE 91/414/EEC CONCERNING THE PLACING OF PLANT PROTECTION PRODUCTS ON THE MARKET

2. TERMS OF REFERENCE

The Scientific Committee on Plants is requested to respond to the following questions in the context of the Commission's work on the implementation of Directive 91/414/EEC[1] concerning the placing of plant protection products on the market.

(1)   What is the relevance for man of the results from the two-year carcinogenicity study in rats?

(2)   Does the available information on dermal absorption allow a reliable estimate to be made for man, if so, at what level?

(3)   Can the Committee comment on the appropriate dietary risk assessment to be used?

(4)   Can it be confirmed that the proposed risk mitigation measures for aquatic organisms would avoid unacceptable risk for the aquatic environment?

(5)   Can it be confirmed that the proposed risk mitigation measures are adequate to protect non-target arthropods and honey bees?

3. BACKGROUND

Esfenvalerate is an existing active substance in the context of Directive 91/414/EEC and is covered by Commission Regulation (EEC) No 3600/92[2] laying down the detailed rules for implementation of the first stage of the work programme provided for in the Directive.

In order to prepare the opinion the Committee had access to documentation comprising a Monograph prepared by Portugal as Rapporteur Member State (RMS), information from the ECCO[3] Peer Review programs and responses[4] from the notifier to the questions[5] from Committee.

Esfenvalerate is a synthetic pyrethroid insecticide and is effective against harmful organisms via contact and stomach action. It is authorised for use in plant protection products in agriculture, horticulture and forestry.

---

[1] OJ L 230, 19. 08.1991, p.1
[2] OJ L 366, 15. 12.1992, p.10
[3] European Community Co-ordination
[4] SCP/ESFEN/10,12,13,14,15,19.
[5] SCP/ESFEN/6

The active substance belongs to the group of pyrethroids acting as contact and stomach poison with neurotoxic effects on insect pests.

Fenvalerate is a racemic mixture of four stereoisomers (isomers ratios: A-alpha, 25%; B-alpha, 25%; A-beta, 25% and B-beta 25%) owing to the presence of two chiral centres. Esfenvalerate is the A-alpha isomer, one of the four chiral isomers and was developed with the knowledge that it was the biologically active component of the fenvalerate racemic mixture. Esfenvalerate contains a minimum of 83% A-alpha isomer together with a maximum of 14% related isomers. Esfenvalerate is classified, according to its structure, as a Type II pyrethroid. Pyrethroids which contain an alpha-cyano-group have been shown to act on the peripheral nervous system as well as the central nervous system.

---

4. OPINION

4.1. Question 1

What is the relevance for man of the results from the two-year carcinogenicity study in rats?

Opinion

Fenvalerate and esfenvalerate were consistently negative when evaluated in a standard battery of genotoxicity tests. The Committee concluded that mammary gland tumours in the Sprague Dawley rats were unlikely to be treatment related. Moreover these effects were not confirmed in another rat strain. Thus these findings are considered to be non relevant for humans.

The Committee concluded that the increase in Testicular Interstitial Cell Tumours (ICT) is probably related to treatment in SLC:Wistar rats. This effect was not evident in two other studies with Sprague Dawley rats. In addition, the Committee noted the remarkable differences in ICT susceptibility between Wistar rats and humans. Accordingly, the Committee concluded that the effect observed in Wistar rats is not considered to be predictive of a carcinogenic potential for man.

4.1.1. Scientific Background on which the Opinion is Based

Genotoxicity

Fenvalerate and esfenvalerate were consistently negative when evaluated in a standard battery of genotoxicity tests.

Carcinogenicity and chronic toxicity

Administration of fenvalerate to male and female B6C3F1 mice and ddY mice resulted in no increase in tumour incidence at any site. Esfenvalerate did not increase the incidence of any tumour type in CD-1 mice[6].

Three studies in rats were submitted:

Lifetime feeding study in rats with SD-43775 (fenvalerate) technical.
Fenvalerate was administered to Sprague Dawley rats at dietary levels of 0 ,1, 5, 25 or 250 ppm in the diet for 2 years. The control group consisted of 183 rats/sex, and the treated groups consisted of 93 rats/sex.
The study was conducted prior to development of GLP[7] and was not conducted in accordance with current procedures of Quality Assurance, but complies to a great extent to the requirements of Directive 87/18/EEC[8]. The main deviations were: ophthalmologic examinations were not performed; the histopathological examinations did not include aorta, oesophagus and femur.
Report AT 81-0182 (1978)

Lifetime feeding study in rats with SD-43775 (fenvalerate) technical.
Fenvalerate was administered to Sprague-Dawley rats (50/sex) at dietary dose levels of 0 or 1000 ppm in the diet for 2 years.
The study was conducted prior to GLP practice, but was conducted in accordance with current procedures of Quality Assurance and complies to a great extent to the requirements of Directive 87/18/EEC. The main deviations were: no interim kills; urinalysis, haematology, clinical biochemistry were performed only at termination; the absence of ophthalmologic examination.
Report AT-91-0231 (1979)

A two-year chronic study of S-5602 (fenvalerate) in rats
Fenvalerate was administered to SLC:Wistar rats (80/sex) at dose levels of 0, 50, 150, 500 and 1500 ppm in the diet for 2 years. No GLP compliance because the study was conducted prior to GLP requirements but was conducted in accordance with current procedures of Quality Assurance and complies to a great extent to the requirements of Directive 87/18/EEC. The main deviations were: urinalysis, haematology, clinical biochemistry and ophthalmology were performed only at termination.
Report AT-10-0278 (1981)

The Committee is of the opinion that the studies were poorly reported but they were considered acceptable in terms of the protocol and quality when compared to the established toxicological protocols used at the time when they were performed. Moreover, the studies were performed with fenvalerate rather than esfenvalerate as would have been preferable in order to allow a more accurate hazard assessment to be made. In the light of the similarity in pharmacokinetics, metabolism and toxicity of these two compounds, the Committee however feels that an adequate assessment can be made in this particular case.

---

[6] Response to questions raised by the Toxicology subgroup of the Scientific Committee on Plants at its meeting held on 16 December 1999. SUMITOMO CHEMICAL COMPANY LTD (ref. SCP/ESFEN/013).
[7] Good Laboratory Practice
[8] OJ L 15, 17.01.1987, p. 29

Mammary gland tumours in Sprague Dawley rats
In the first chronic exposure study in Sprague Dawley rats. Report AT 81-0182 (1978), fenvalerate treatment resulted in a non-dose related increase in benign mammary gland tumours in females. No increase in mammary gland tumours was seen in a second study Report AT-91-0231 (1979) conducted at a dose level of fenvalerate higher than that used in the first study.

The Committee did not consider it appropriate to combine together the two studies since they were carried out in different periods and comparisons of results would be complex due to the fact that some aspects of the two experimental protocols are different.

In the first chronic exposure study in Sprague Dawley rats (Report AT 81-0182, 1978) statistically significant differences were seen in the number of benign mammary gland tumours at 5 and 250 ppm in the interim death group, in the number of the total (benign and malignant) mammary gland tumours at study termination in the 25 ppm group, and in combined tumours (interim and terminal deaths) at 250 ppm. The increases in benign, malignant or total mammary gland tumours were not dose related (Peto and Cochran-Armitage trend tests were negative) and no significant trend was found. Since these increases were restricted to benign tumours, which occur naturally at high spontaneous incidence in ageing Sprague-Dawley rats, the increases were unlikely to have been caused by the treatment. In addition, there were no indications that treatments were associated with an earlier occurrence of tumours. The occurrence of tumours was within the historical control range (46-65%) for the Sprague Dawley rats (in females frequencies as high as 85% have been recorded). The mammary gland tumours in the control groups ranged from 35% in the first study up to 52% in the second one. In addition, the top dosed group in the first study had an incidence of 50%.

No increases were observed in study Report AT-91-0231 (1979) at the single and higher dietary dose level of 1000 ppm.

The Committee concluded that the weight of the evidence indicates that mammary gland tumours in the Sprague Dawley rats were very unlikely to be treatment related. Moreover these effects were not confirmed in another rat strain (SLC: Wistar rats – Report AT-10-0278 (1981)). Thus these findings are considered to be non- relevant for humans.

Testicular Interstitial Cell Tumours (ICT) in SLC:Wistar rats
It is difficult to make a pathological distinction between ICTs and Interstitial Cell (IC) hyperplasia on morphological grounds. There is a continuum, the only distinguishing feature is the size of the lesions. The testicular ICT data showed a significant positive trend when analysed by the Peto Analysis and the Cochran- Armitage Trend Test. In addition, by using pair-wise comparisons the incidence of these tumours was shown to be significantly increased at 50, (but not at 150 ppm) 500 and 1500ppm in the terminally sacrificed animals when analysed by Fishers' exact test and the Chi-square test. The incidence of these tumours was also shown to be significantly increased at 500 and 1500 ppm in the inter-current sacrifice animals when analysed by Chi-square test. However, it has to be emphasised that the effect was essentially an enhancement of the lesions' size rather than an actual increase in the number of lesions.

No increase in the incidence of ICTs was observed in two studies on Sprague Dawley rats (Reports AT 81-0182 (1978) + AT-91-0231 (1979)).

It has been well established that in the rat chronic elevation of LH[9] (the main trophic hormone that stimulates testosterone production in the testes) can stimulate mitogenesis which can lead to IC hyperplasia and eventually to the formation of benign ICT. A mechanistic study was performed to investigate the hypothesis that chronic exposure to fenvalerate and esfenvalerate may lead to increases in LH and testosterone serum levels. This study failed to demonstrate any significant increase in the levels of the two hormones and failed to provide a mechanistic link between fenvalerate exposure, elevated LH and testosterone levels and the increase in ICT observed in SLC: Wistar rats.

The spontaneous incidence of ICTs is highly variable among different rat strains. The background incidence in the SLC:Wistar rats used in the chronic fenvalerate study can be as high as 93%. ICTs are much less frequent in other rat strains and rare in humans. Moreover there is no epidemiological evidence that humans treated with drugs known to induce ICT in rodents have increased susceptibility to ICT formation.

The Committee concluded that the increase of ICTs is probably related to treatment in Wistar rats. This effect was not evident in the two other studies with Sprague Dawley rats. Thus these findings are considered to be non-relevant for humans.

4.2. Question 2

Does the available information on dermal absorption allow a reliable estimate to be made for man, if so, at what level?

Opinion

The *in vitro* study on the rat and human skin indicates an unusually large difference in dermal absorption (44% versus 0.6%) compared with similar experiments carried out on other active substances. In the absence of more conclusive data the Committee is of the opinion that a more conservative estimate of operator absorption is advisable. To confirm the reliability of such a very low value, a study involving the monitoring of workers in the field would be necessary.

4.2.1. Scientific Background on which the Opinion is Based

Rat and human skin penetration was compared in an *in vitro* test. The study showed a much higher penetration in rat (44%) than in human skin (0.6%). The value obtained for human skin absorption was then used to predict the operator exposure (absorption) level.

The conclusion that human skin absorption rate is low appears to be justified, however the calculated rate should be used with caution to predict operator exposure because the *in vitro* experimental conditions do not necessarily represent the real conditions of exposure *in vivo*. The SCP is of the opinion that *in vitro* experiments used to predict operator exposure absorption should be considered to be at most indicative of an order of magnitude of absorption. When an unusually large difference is observed between dermal absorption between rat and human skin *in vitro* as in this case, it is not prudent to assume that such a difference would actually occur in the

---

[9] Luteinizing hormone

in vivo situation in the field. In order to confirm a value as low as 0.6%, a study involving the monitoring of workers in the field would be necessary.

4.3. Question 3

Can the Committee comment on the appropriate dietary risk assessment to be used?

Opinion

Esfenvalerate belongs to the group of pyrethroids containing an α-cyano-group which are known to be potentially neurotoxic. In addition to a long-term dietary intake risk assessment, as routinely carried out for plant protection products, esfenvalerate should also undergo a short-term acute dietary risk assessment due to its potential neurotoxicity properties.

An ARfD[10] would be needed for this reason.

For guidance on establishing an ARfD, the Committee refers the reader to the "Opinion of the Scientific Committee on Plants on the general criteria for setting acute reference doses for plant protection products", expressed on 28 January 2000[11]. In addition, attention is drawn to the "Report of the International Conference on Pesticide Residues Variability and Acute Dietary Assessment", 1-3 December 1998, York, and the JMPR Report 1998 (FAO PLANT PRODUCTION AND PROTECTION PAPER 148).

4.4. Question 4

Can it be confirmed that the proposed risk mitigation measures for aquatic organisms would avoid unacceptable risk for the aquatic environment?

Opinion

The width of the buffer zones proposed by the Notifier are not considered to be sufficient to avoid unacceptable risk for the aquatic environment because they do not adequately take into account effects on sensitive aquatic taxa. Although higher-tier mesocosm studies were performed as part of the risk assessment, it is the Committee's opinion that the Notifier's interpretation of the results of these studies is inadequate and does not take into account all relevant effects.

4.4.1. Scientific Background on which the Opinion is Based

The risk mitigation measures proposed to avoid unacceptable risk for the aquatic environment involve drift-reducing application techniques and the establishment of buffer zones. The conclusion of the Evaluation Group was that risk mitigation measures can be defined at the Member State level and that safe uses of esfenvalerate have been demonstrated.

---

[10] Acute Reference Dose
[11] http://europa.eu.int/comm/dg24/health/sc/scp/out02_ppp_en.html

The Notifier proposed the following buffer zones (all are in units of meters and are taken from Tables 17 & 18 Doc. 7753/VI/97-app.2):

Crop	TER[12]=1, Northern Europe	TER=1, Southern Europe	TER=1, Overall	TER=10, Overall
Orchards	10	10	10	40
Vineyards	5	10	10	20
Field/arable	1	2	2	10
Cotton				15
Kohlrabi, cauliflower, broccoli				3

These were based on the ratio of mesocosm NOEC[13]/initial PEC[14]$_{SW}$ and show cases for which an application factor of 1 or 10 is applied to the NOEC data. The NOECs employed in the calculation were for *Daphnia* (0.20 µg/L) and fish (0.25 µg/L), which were not the most sensitive endpoints in the mesocosm studies. For example, Lozano et al. (1992) found 50% reductions in many macroinvertebrate taxa at 0.01-0.08 µg/L. The most sensitive species in the mesocosm studies were affected at the lowest concentrations tested (hence no NOEC could be determined) and at concentrations 20 times lower than those affecting *Daphnia* or fish. It should be emphasised that these effect concentrations are in the range of the PEC$_{SW}$ values associated with the 'best case' usage of esfenvalerate (Table 5, 7753/VI/97-app 2). Aquatic insects and some of the copepods were the most sensitive invertebrate taxa in these mesocosms and showed little or no recovery at any of the concentrations over the 8-week study period. This study also indicated persistence of esfenvalerate in the sediment, possibly leading to delayed effects on the benthos (1 and 10 µg/g in sediment after 1st & 2nd applications, respectively). There were no data provided in the monograph with regard to esfenvalerate's effects on sediment-dwelling organisms per se, which given its physical/chemical properties is rather surprising. However, this mesocosm study suggests that benthic invertebrates could be exposed to a chronic risk from esfenvalerate.

Two additional studies cast doubt on the adequacy of the recommended buffer zones. Fairchild et al. (1992) found reductions in zooplankton and benthic macroinvertebrates to occur at 0.25 µg/l, which was the lowest concentration tested in their study, and therefore a NOEC could not be determined for these groups. This study also included laboratory toxicity tests which found bluegill 96 h LC$_{50}$[15]=0.31 µg/L (95%CL=0.25-0.40) and *Daphnia* 48 h LC$_{50}$=0.27 µg/L (95% CL=0.19-0.42). These acute laboratory LC$_{50}$s (without any application factors applied) are very close to the mesocosm NOECs used by the Notifier to calculate buffer zones. Also, Webber et al. (1992) detected effects on insect emergence and reduced zooplankton nauplii at measured esfenvalerate concentrations of 0.18 µg/L.

In summary, all of the above studies provide evidence that employing NOEC values in the range of 0.2-0.25 µg/L is likely to underestimate effects on aquatic species. The Committee disagrees with the Notifier's interpretation of the mesocosm studies on two counts: 1) the Notifier concluded that impacts on aquatic organisms arising from very high, multiple esfenvalerate inputs

---

[12] Toxicity Exposure Ration
[13] No Observed Effect Concentration
[14] Predicted Environmental Concentration Surface Water
[15] Lethal concentration, median

were generally minimal, and 2) the Notifier concluded that aquatic ecosystems proved to be remarkably resilient to repeated, high-level applications of esfenvalerate. In fact, effects were demonstrated on a number of invertebrate groups in the mesocosm studies, and only a few of the studied taxa showed clear signs of recovery (see p. 13, para 2-3 on recovery).

One of the intentions of higher-tier risk assessments, such as mesocosm studies, is to include a broader range of species than is tested during the initial risk assessment. Therefore the Committee feels it is inappropriate for the Notifier to focus interpretation of the mesocosm studies only on fish and cladocerans and to ignore the adverse effects on the other taxa detected in these studies. The buffer zones calculated by the Notifier, based on toxicity to Cladocera and fish, would not necessarily be sufficient to protect more sensitive groups of invertebrates.

The RMS noted that there is no clear guidance available on TER values calculated from mesocosm studies. In the Committee's opinion it may be scientifically defensible to use a TER of 1 for a mesocosm or field study if: 1) it can be demonstrated that the design is environmentally realistic in terms of the key features controlling the fate and effects of the chemical under study and 2) if it can be shown that the design (including the selection of endpoints) is of sufficient statistical power that biologically relevant effects will be detectable.

4.4.2. REFERENCES

Fairchild JF, La Point TW, Zajicek JL, Nelson MK, Dwyer FJ, Lovely PA. 1992. Population-, community-, and ecosystem-level responses of aquatic mesocosms to pulsed doses of a pyrethroid insecticide. Environ. Toxicol. Chem. 11: 115-129.

Lozano SJ, O'Halloran SL, Sargent KW, Brazner JC. 1992. Effects of esfenvalerate on aquatic organisms in littoral enclosures. Environ. Toxicol. Chem. 11: 35-47.

Webber EC, Deutsch WG, Bayne DR, Seesock WC. 1992. Ecosystem-level testing of a synthetic pyrethroid insecticide in aquatic mesocosms. Environ. Toxicol. Chem. 11: 87-105.

4.5. Question 5

Can it be confirmed that the proposed risk mitigation measures are adequate to protect honey bees and other non-target arthropods?

Opinion

Overall, the data show that the intended uses of esfenvalerate pose an unacceptable risk to non-target arthropods under Annex VI criteria, unless specific risk mitigation measures are implemented. No risk mitigation measures for honey bees or other non-target arthropods were proposed. Specific risk mitigation measures that should be considered for honey bees include: (a) the control of the timing of application (time of day and season) with respect to (i) honey bee activity and (ii) flowering of the crop, and (b) restrictions on spraying close to hives. Specific risk mitigation measures that should be considered for other non-target arthropods include restrictions on the number of applications per season, restrictions on spraying close to field margins, selection of appropriate application techniques to minimise spray drift, and limits on the extent of application.

The data show that while esfenvalerate has high direct contact and oral toxicity to honey bees, the observable effects of esfenvalerate are much reduced under field and semifield conditions. On the basis of this data, the SCP is generally satisfied that the use of esfenvalerate will not pose an unacceptable risk to honey bees, so long as the active substance is applied at concentrations of 30g as/ha or less and the above risk mitigation measures can be implemented. However, the SCP notes that the proposed application rates of the compound in orchards and vineyards is in some cases several times higher than has been investigated in field and semifield trials. It therefore recommends that the use of this active substance at these high application rates should be supported by higher tier testing in the field.

Unfortunately there were relatively few laboratory and semifield tests to estimate the toxicity of esfenvalerate to other beneficial arthropods. However, data on two full field trials, both conducted on winter wheat, were also submitted. The field trials showed that some taxa exhibited a statistically significant decline in numbers compared to control, but these differences were temporary, in all likelihood mediated by recolonisation from untreated areas. The SCP believes that in principle, it is entirely appropriate to take natural recolonisation from off-crop habitats into account in the risk assessment process. However, initial population effects were observed. Furthermore, the process of recovery through immigration will depend on the usage pattern of the compound. Thus, there remains a need for specific risk reduction measures (see above).

Given the apparent reduction of *Aphidius* in one of the field trials (which the notifier has argued is not treatment related), and the importance of parasitoids as natural enemies, the SCP feels that the Notifier should conduct a laboratory test on this taxon. Furthermore, the SCP agrees with the RMS that the use of esfenvalerate in orchards and vineyards should be supported by higher tier testing in the field. This is particularly important given the high doses used in these crops, and the fact that the main field trials have only dealt with esfenvalerate use in cereals. The proposed high rates of application in forests and nurseries also merit further consideration.

4.5.1. Scientific Background on which the Opinion is Based

(a) Honey Bees

Review of available ecotoxicity data

Details of a number of tests, using two principal formulations (EC[16] and SC[17]), were supplied by the notifier.

Laboratory tests
The laboratory tests indicate that esfenvalerate exhibits high contact toxicity to honey bees (48 hr $LD[18]_{50}$ 0.06-0.07 µg as / bee) and high oral toxicity (LD_{50} 0.21 - 0.8 µg as / bee).

Semifield trials
The mortality from residual exposure was estimated in several cage and semifield trials. For example, 36% bee mortality was observed after 2 days following applications to oil seed rape in cages at 50 mg/l. Thirteen percent mortality was observed in bee colonies exposed for 24 hours to flowering rape samples that were sprayed 8 hours before at 30 mg/l. Unreplicated cage tests

---

[16] Emulsifiable Concentrate
[17] Suspension concentrate
[18] Lethal Dose, median

were conducted in Germany in which flowering *Phacelia* plots containing bee colonies were sprayed at certain times (midday or evening) with 25 g as / ha. The mortality in the test colonies were comparable to pre- and post- treatment levels and the water controls, while foraging activity was reduced by about 50% up to 3 hours after application in the midday treatments. A similar test was conducted in France whereby bee colonies were confined to tunnels containing flowering white mustard. Application rates of 15 g as/ ha and 30 g as/ha had no discernible effect on bee mortality but there was evidence of repellence that lasted 1-5 hours depending on concentration. Additional tests were also conducted that included a high dose of esfenvalerate (60 g as /ha), with similar results. A bee brood feeding test in which 1.5g as/l was provided in syrup to 3 separate colonies found an initial high level of mortality in treated colonies which appeared to last approximately a week (2325 dead bees overall compared to 317 in control colonies), but there were no detectable effects on brood production (which was highly variable).

Field trials
Field trials conducted in Hungary found that mortality was not significantly higher in bee colonies situated next to a field of flowering winter rape that were sprayed with 15 g as/ha (in the evening), compared to similar controls colonies. However, there appeared to be a small reduction in the number of foraging visits on the first morning after treatment.

Importance and adequacy of risk mitigation measures

Given the high sensitivity of bees reported under laboratory conditions, risk mitigation measures should be implemented before safe use can be confirmed. No specific risk mitigation measures for honey bees have so far been proposed that the Notifier wishes to support through Annex 1. However, measures that should be considered include: (a) the control of the timing of application (time of day and season) with respect to (i) honey bee activity and (ii) flowering of the crop, and (b) restrictions on spraying close to hives.

The data show that while esfenvalerate has high direct contact and oral toxicity, the observable effects of esfenvalerate are much reduced under field and semifield conditions. In general, tests were conducted at appropriate concentrations, given the intended uses. As the RMS points out, cage and tunnel tests represent relatively severe tests because bees are kept in relative close proximity to the sprayed crop. On the basis of this data, the SCP proposes that, if the above risk mitigation measures are implemented, then applications up to 30 g as/ha will not pose an unacceptable risk to honey bees.

The SCP notes that apple and pear orchards, which support honey bees, may require regular (1-5) application rates of up to 182 g as /ha (although there is some discrepancy between tables). This concentration is several times the maximum dose investigated in field and semifield trials (60 as/ha). If the product were applied at these concentrations, then it would extremely difficult to predict the risk to bees under these conditions. The SCP therefore proposes that the use of this compound at these rates in orchards and vineyards, or indeed any crop, should be supported by higher tier testing in the field.

(b) Other arthropods

Review of available ecotoxicity data

Laboratory and semifield tests
Unfortunately there were relatively few laboratory tests to estimate the toxicity of esfenvalerate to other beneficial arthropods. The laboratory tests presented by the Notifier included only a lacewing species (*Chrysoperla carnea*), 2 species of linyphiid spider and a carabid beetle (*Poecilius cupreus*). No tests on a predatory hoverfly, a predatory mite, a folliage dwelling beetle or a hymenopteran parasitoid were originally presented. However, the Notifier has since provided details of an extended laboratory study on the effects of esfenvalerate on the predatory mite *Typhlodromus pyri*. Esfenvalerate sprayed onto glass plates at a rate of 12.5 g as /ha killed 100% of linyphiid spiders after 48 hrs, compared to 9.5% control mortality. Glass plates sprayed at a rate of 4.71 g as /ha produced 90.5% mortality in lacewing larvae compared to 26% control mortality and there was some evidence for a small reduction in the fecundity of treated survivors. A separate semifield trial found that lacewing larvae (*Chrysoperla carnea*) on pot grown bean plants showed a slight, but not significant, reduction in population size when pots were sprayed with fenvalerate and esfenvalerate at rates of 30 and 12.5 g as/ha respectively. Applications of 12.5g as/ha on quartz sand caused only 3.3% mortality in *Poecilus cupreus* compared to 0% controls and, while treated beetles showed initial symptoms of toxic effects, rates of feeding activity were comparable. When *Typhlodromus pyri* were exposed to leaves treated with 0.015-0.15 g as/ha for two weeks, mean mortality was higher and reproductive rate was lower than controls, with overall effects estimated as 10-90.7 % for the range of concentrations.

Field trials
Data from a field trial involving an application of 5g as/ha, on winter wheat was originally presented, but data from a field study on winter wheat using applications of 7.5 g as/ha and 15g as/ha 20 days apart have been submitted only recently. The earlier study employed replicated (3) plots, and found no statistically significant reductions in population size of any taxon. However some groups did show marked reductions (e.g. 47% reductions of coccinelid larvae relative to control) and the variance in the data appeared high. The second field trial at higher dose rates found significant reductions in linyphiids and aerial fauna including *Aphidius*, although differences between treatment and controls were not significant towards the end of the study. Staphylinid beetles showed some significant reductions in numbers, but carabids were not significantly reduced.

Importance and adequacy of risk mitigation measures

Given the apparent reduction of *Aphidius* in the field trial (which the Notifier has argued is not treatment related), and the importance of parasitoids as natural enemies, the SCP feels that it would be appropriate to conduct a laboratory test on this taxon. While the magnitude and duration of effects of esfenvalerate on arthropod species in field trials appeared limited, the observed recovery was likely to be strongly influenced through recolonisation of these species from untreated areas. Given that there were observed short-term reductions of beneficial beetle, spider and parasitoid species in field trials, and the fact that that the permanent viability of source populations will depend on the usage pattern of the compound, then there remains a need for specific risk reduction measures.

Thus, the data show that the intended uses of esfenvalerate pose an unacceptable risk to non-target arthropods under criteria in Annex VI, unless specific risk mitigation measures can be implemented. In the following paragraphs we expand on why we consider risk mitigation to be important even in the light of the observed recovery, and we use the same reasoning to identify some appropriate mitigation measures.

The observation of population recovery through immigration from unsprayed sources raises a fundamental issue: should it be incorporated into the risk assessment process, and if so how? Furthermore, if recovery is considered, what risk mitigation measures should be implemented to ensure that recovery can take place via this process? Immigration is a natural ecological process, and as such, the SCP feels that it is appropriate to consider it in the context of a risk assessment. Indeed, it would often be impractical to attempt to rule it out as a factor in field trials. However, it is important to recognise that the actual rates of immigration of arthropods into treated areas are likely to be highly dependent on the sizes and proximities of suitable source populations. Thus, repeated applications of the chemical may eventually deplete the sizes of the source populations through continued attrition (Sherratt & Jepson 1993). Similarly, the rates of recovery of arthropod populations within the treated crop will depend on the actual sizes of populations that were lost after treatment. Thus, if sprays are applied extensively, then large populations are likely to be affected and the subsequent rate of recovery is likely to be low. This reasoning is supported by experimental data which show that the rate of recovery will depend on the area of crop sprayed (e.g. Jepson & Thacker 1990 ; Thomas *et al.* 1990). Another important caveat is that the rate of recovery of arthropods in the treated area will depend not just on the frequency and extent of application of the pesticide in question, but on the suitability of the surrounding habitats for arthropods, and the toxicities and patterns of use of other pesticides.

Whenever initial population reductions are observed in the field, then this should raise cause for concern. Furthermore, whenever immigration is seen as an important factor in the recovery process, then it is likely that the usage pattern of the compound will have a correspondingly high influence on long-term viability of affected populations in the treated area. Therefore, in such cases, it is necessary to implement risk mitigation measures. One appropriate measure is to restrict spraying close to any off-crop areas that are likely to support significant populations of beneficial arthropods. Such a restriction might also involve selection of appropriate application techniques to minimise spray drift. While it is also recognised that repeated or extensive applications of the compound may affect the ability of beneficial arthropod populations to recover, there are currently no agreed guidelines with which to set quantitative limits on these parameters. Given the uncertainty, we recommend that selection of appropriate upper levels of frequency and extent of application should be based on a consideration of the conditions under which field trials were conducted.

As far as we can assess, the majority of critical GAP[19] scenarios provide a considerable interval outside a short period of spraying activity to enable recovery, if it occurs at approximately the observed rates. The likely effects of esfenvalerate and the subsequent recovery of beneficial arthropods under certain GAPs, are however difficult to estimate. As noted in section 2.1.2, the SCP agrees with the RMS that the use of esfenvalerate in orchards and vineyards should be supported by higher tier testing in the field and notes that a field trial was planned for 1999 in the south of France. The proposed high rates of application in forests and nurseries (up to 125 g as/ha in two applications) also merit further consideration.

---

[19] Good Agricultural Practice

4.5.2. REFERENCES

Jepson, P.C. & Thacker, J.R.M. (1990). Analysis of the spatial component of pesticide side-effects on non-target invertebrate populations and its relevance to hazard analysis. *Functional Ecology*, **4**, 349-355.

Sherratt T.N. & Jepson, P.C. (1993). A metapopulation approach to modelling the long-term effects of pesticides on invertebrates. *Journal of Applied Ecology*, **30,** 696-705.

Thomas, C.F.G., Hol, E.H.A., Everts, J.W. (1990). Modelling the diffusion component of dispersal during recovery of a population of linyphiid spiders from exposure to an insecticide. *Functional Ecology*, **4**, 357-368.

5. ACKNOWLEDGEMENTS